Editorial

Ethnicity and Stroke
Beware of the Fallacies

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The role of ethnicity in stroke has been the subject of a considerable number of published reports. A quick Medline search detected 454 citations on “ethnicity and stroke,” 386 on “stroke in blacks,” 251 on “stroke in African Americans,” and 74 on “stroke in Hispanics,” of which only a few can be mentioned here.1-12 There even exists a journal dedicated to ethnicity and health.13

However, the assumption that ethnicity is an isolated epidemiological variable delineating clinically distinct disease subgroups is controversial.14-17 The very concept of the word may be confounded with race (“black”), a common language or culture (“Hispanic”), a shared geographic origin (“Asian”), or a presumed common descent with diffuse boundaries (“Caucasian”). Ethnic categories are usually not defined in scientific reports, which results in dubious findings that are difficult to compare.18 The idea that a socially defined variable may reveal biological differences is fallacious, leading dangerously to biological determinism.15 For example, the genetic variation between races, traditionally classified on phenotype, is only slightly greater (10%) than that between nations (6%), and much larger within a local population (84%).19,20 Moreover, the genes responsible for skin color are few and are not associated with genetic markers for disease.14

Ethnicity as a variable may be too greatly influenced by cultural attitude and therefore biased. In the past, this attitude led to the entire invention of diseases on the basis of race. At a time when the genetic inequality of races was considered obvious, the existence of these diseases was not questioned.15 In the present, ethnicity may be used euphemistically to avoid racist implications. A survey of 48 medical schools in the United States revealed that up to 91% of clerkship directors answered “yes” or “variable” after being queried whether students were taught by example to use the terms “black” or “white” when introducing case presentations.20 In another study, “black” patients were far more likely than “whites” to be racially identified at morning report.21 As recently as 1991, arterial hypertension has been related to skin color, even allowing for the fact that darker “blacks” may as a consequence be poorer and suffer more psychosocial stress.22

Ethnic classification may vary from one community to another, as the perception of an ethnic group may be different across countries. As Caldwell and Popeno20 put it, “what is black to someone from the United States may be white to a Brazilian or a Caribbean islander.” It may be added that the authors of the present editorial, both of European descent and born and raised in Spanish-speaking countries, would probably be classified as “Hispanic” in the US, although neither is of Spanish descent. Obviously, there is no such thing as a “Hispanic” ethnic group in Spain or Latin America.

Ethnicity is not a dichotomous variable, such as gender. How black is black? How is a person classified whose father is “white” and mother “black”? What about “mixed” grandparents? How does one classify a phenotypically “black” (by US standards) Spanish-speaking national from Central America? How are his children classified? Finally, how white is white? Do a Scot from Edinburgh and an Italian from Milan belong to the same ethnic group?21,27

Ethnicity has been shown to vary through time. One study23 showed an increase in nonwhite races on death records compared with birth records in the same population and indicated that this reclassification may have been politically biased. A study on infant mortality24 showed that 87.3% of infants were ethnically reclassified at death, with the greatest inconsistency involving “Hispanics” (30.3%) and the least “whites” (1.2%).

To avoid the shortcomings linked to classification, it has been proposed (and is now used in many reports) that patients entering population studies “self-classify” their ethnicity,25 assuming that “racial and ethnic categories are understood by the populations questioned.”26

However, misinterpretation, confusion, and self-reclassification have been found in these cases.26,27 One striking example is that the category “South and Central American” was thought by respondents in one census to refer to natives of the south and central United States!26

To define disease on the basis of biologically determined ethnicity might perhaps hide socioeconomic differences that are the real cause of disease in some ethnic groups. Affluent “blacks” from the US East Coast may not necessarily develop the same disease patterns as poor inhabitants from a comparable group in the southern states. Many reports on interethnic differences are not adjusted for socioeconomic variables, and this is perhaps the most important bias involving ethnicity research.

Senior and Bhopal14 have proposed that a “sound epidemiological variable” should be accurately measured and should differentiate populations by some underlying characteristic relevant to health (such as income, childhood circumstances, hormonal status, genetic inheritance, and lifestyle).
The observed differences in patterns of disease should generate testable etiologic hypotheses or be applicable to the planning and delivery of health care. However, these observed differences have rarely led to applicable hypotheses beyond the initial observation, as researchers have emphasized the understanding of etiology rather than the development of specific health policies.

Because ethnicity is difficult to define, it is consequently difficult to measure. Ethnic populations are more and more heterogeneous, and ethnic heterogeneity is often underestimated. An example are the well-conducted studies of coronary heart disease and stroke in “Japanese” men living in Japan and in “Japanese-American” men from Hawaii and California (the Ni-Hon-San studies), which showed a gradient from high stroke rates in Japan to low rates in California and a reverse trend for coronary heart disease. More than 11,000 men were included. These theoretically conflicting trends could not be explained only by conventional risk factors or blood pressure distributions, “although the subjects shared a common ethnic background.” It is very probable that ethnic heterogeneity is as important a confounder to the findings as environmental, dietary, and various other factors. The highly intriguing results of such studies, rather than being dismissed, merit a more rigorous approach.

Genetic research may be one way of achieving this. For instance, in the late eighties, familial cavernous malformations were reported to be more common among “Hispanics.” However, more precise research showed linkage of these cavernous malformations to chromosome 7p13–15 and 3q25.2–27. Of malformations may show the same or other linkages, for instance, in the late eighties, familial cavernous malformations were reported to be more common among “Hispanic” men from Hawaii and California (the Ni-Hon-San studies), which showed a gradient from high stroke rates in Japan to low rates in California and a reverse trend for coronary heart disease.

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The consequences of flawed ethnicity research may lead to the assumption that ethnic minorities are an unhealthy social burden, that there are “ethnic” diseases which separate specific groups from the general population, that consequently they do not merit any further attention, and that “whites” are the “gold standard” of health. All this could do nothing but fuel racial prejudice.

Patterns of disease that distinguish different ethnic groups may well exist and should be adequately established if better health standards for the populations concerned can be achieved as a consequence. Failure to do so may just be an example of “black box epidemiology,” resulting in futile and never-ending reports on interethnic differences that have been likened to repeatedly punching a soft pillow.

References


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